Cohesin and CTCF differentially affect chromatin architecture and gene expression in human cells.

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Public Summary:

Scientific Abstract:
Recent studies of genome-wide chromatin interactions have revealed that the human genome is partitioned into many self-associating topological domains. The boundary sequences between domains are enriched for binding sites of CTCC-binding factor (CTCF) and the cohesin complex, implicating these two factors in the establishment or maintenance of topological domains. To determine the role of cohesin and CTCF in higher-order chromatin architecture in human cells, we depleted the cohesin complex or CTCF and examined the consequences of loss of these factors on higher-order chromatin organization, as well as the transcriptome. We observed a general loss of local chromatin interactions upon disruption of cohesin, but the topological domains remain intact. However, we found that depletion of CTCF not only reduced intradomain interactions but also increased interdomain interactions. Furthermore, distinct groups of genes become misregulated upon depletion of cohesin and CTCF. Taken together, these observations suggest that CTCF and cohesin contribute differentially to chromatin organization and gene regulation.

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